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DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ

<input type="checkbox"/>	L14	l8 and (fibronectin near2 culture)	2
<input type="checkbox"/>	L13	L12 and l9	1
<input type="checkbox"/>	L12	L11 same l10	6
<input type="checkbox"/>	L11	collagen type I or type I collagen or collagen typeI or typeI collagen	7275
<input type="checkbox"/>	L10	cd14 same cd34 same cd45	439
<input type="checkbox"/>	L9	L7 and mesenchymal	573
<input type="checkbox"/>	L8	L7 and multipotent	73
<input type="checkbox"/>	L7	monocyte near2 differentiation	1968

DB=USPT,PGPB; PLUR=YES; OP=ADJ

<input type="checkbox"/>	L6	KODAMA-HIROAKI!	16
<input type="checkbox"/>	L5	KUWANA-MASATAKA!	2

DB=USPT; PLUR=YES; OP=ADJ

<input type="checkbox"/>	L4	6274378.pn.	1
<input type="checkbox"/>	L3	6465247.pn.	1
<input type="checkbox"/>	L2	6465247.pn.L1	0
<input type="checkbox"/>	L1	6534055.pn.	1

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FILE 'MEDLINE' ENTERED AT 17:00:02 ON 17 JUL 2007

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=> s monocyte ⁽¹⁾same multipotent ← m
L1 0 MONOCYTE SAME MULTIPOTENT

=> s monocyte and multipotent
L2 418 MONOCYTE AND MULTIPOTENT

=> s l2 and fibronectin
L3 4 L2 AND FIBRONECTIN

=> s monocyte and osteoblast
L4 1275 MONOCYTE AND OSTEOBLAST

=> s monocyte and mesenchymal
L5 1211 MONOCYTE AND MESENCHYMAL

=> s l5 and fibronectin
L6 54 L5 AND FIBRONECTIN

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 26 DUP REM L6 (28 DUPLICATES REMOVED)

=> s l7 and py<2004
1 FILES SEARCHED...
4 FILES SEARCHED...
L8 17 L7 AND PY<2004

=> s l8 and py<2003
2 FILES SEARCHED...
L9 14 L8 AND PY<2003

=> disp l9 ibib abs 1-14

L9 ANSWER 1 OF 14 MEDLINE on STN
ACCESSION NUMBER: 97459730 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9315690
TITLE: Integrin-mediated stimulation of monocyte
chemotactic protein-1 expression.
AUTHOR: Marra F; Pastacaldi S; Romanelli R G; Pinzani M; Ticali P;
Carlioni V; Laffi G; Gentilini P
CORPORATE SOURCE: Istituto di Medicina Interna, Universita di Firenze,
Florence, Italy.. f.marra@dfc.unifi.it
SOURCE: FEBS letters, (1997 Sep 8) Vol. 414, No. 2, pp.
221-5.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands

DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 5 Nov 1997
Last Updated on STN: 5 Nov 1997
Entered Medline: 21 Oct 1997

AB We investigated whether activation of integrin receptors could modulate the expression of monocyte chemotactic protein-1 (MCP-1) in human hepatic stellate cells (HSC), mesenchymal cells responsible for extracellular matrix synthesis within the liver. When compared to non-adherent cells, HSC plated on collagen types I or IV, or fibronectin, showed increased MCP-1 gene expression and protein secretion in the conditioned medium. Increased MCP-1 secretion was also observed when cells were plated on dishes coated with a monoclonal antibody directed against the beta1-integrin subunit, demonstrating that ligation of beta1-integrins is sufficient to stimulate MCP-1 expression. Conversely, integrin-independent cell adhesion on poly-L-lysine did not modify MCP-1 secretion. Disruption of the actin cytoskeleton by cytochalasin D blocked the collagen-dependent increase in MCP-1 secretion. Chemotactic assay of HSC-conditioned medium showed that HSC plated on collagen secrete higher amounts of chemotactic factors for lymphomonocytes, and that MCP-1 accounts for the great majority of this effect. These findings indicate a novel mechanism of MCP-1 regulation possibly relevant in those conditions where HSC interact with an altered extracellular matrix.

L9 ANSWER 2 OF 14 MEDLINE on STN
ACCESSION NUMBER: 93112431 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8417761
TITLE: In vitro effects of pentoxifylline on smooth muscle cell migration and blood monocyte production of chemotactic activity for smooth muscle cells: potential therapeutic benefit in the adult respiratory distress syndrome.
AUTHOR: Kullmann A; Vaillant P; Muller V; Martinet Y; Martinet N
CORPORATE SOURCE: INSERM U. 14, CLERC Poumon, CHU, Nancy-Vandoeuvre, France.
SOURCE: American journal of respiratory cell and molecular biology, (1993 Jan) Vol. 8, No. 1, pp. 83-8.
Journal code: 8917225. ISSN: 1044-1549.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199302
ENTRY DATE: Entered STN: 19 Feb 1993
Last Updated on STN: 19 Feb 1993
Entered Medline: 3 Feb 1993

AB The adult respiratory distress syndrome (ARDS) is a severe lung condition characterized by an acute lung injury leading to a massive intra-alveolar fibrosis with rapid lung failure. ARDS intra-alveolar fibrosis results from the migration of mesenchymal cells (mainly smooth muscle cells [SMC]) into the alveoli through alveolar epithelial basement membrane gaps resulting from the injury. SMC migration is followed by their replication and production of extracellular matrix, which leads to fibrosis. Thus, any pharmacologic agent able to prevent SMC migration should prevent, at least in part, intra-alveolar fibrosis. SMC migration is thought to be due to the presence, in the alveolar spaces, of chemotactic factors for mesenchymal cells, such as fibronectin and platelet-derived growth factor (PDGF). The local presence of these chemotactic factors can be due to plasmatic leakage,

platelet degranulation, and mononuclear phagocyte activation. Pentoxifylline is a methylxanthine interacting with the biology of several types of cells, including red blood cells, neutrophils, blood monocytes, and endothelial cells. Pentoxifylline prescription has been suggested in ARDS with respect to its activity on neutrophils, its inhibition of tumor necrosis factor-alpha (TNF) release by mononuclear phagocytes, and its prevention of TNF-induced lung injury. Since pentoxifylline can modulate the migration of several cell types, we hypothesized that it could interfere with mesenchymal cell migration. SMC migratory response was measured in vitro with modified Boyden chemotactic chambers in the presence of PDGF, fibronectin, "platelet extract," and activated blood monocyte supernatants. Pentoxifylline, at therapeutic levels, significantly reduced SMC migration in response to the presence of these chemotactic activities. (ABSTRACT TRUNCATED AT 250 WORDS)

L9 ANSWER 3 OF 14 MEDLINE on STN
 ACCESSION NUMBER: 90118782 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2609937
 TITLE: Gliosarcoma: an immunohistochemical study.
 AUTHOR: Grant J W; Steart P V; Aguzzi A; Jones D B; Gallagher P J
 CORPORATE SOURCE: Institut fur Pathologie, Universitats-Krankenhaus, Zurich, Switzerland.
 SOURCE: Acta neuropathologica, (1989) Vol. 79, No. 3, pp. 305-9.
 Journal code: 0412041. ISSN: 0001-6322.
 PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199002
 ENTRY DATE: Entered STN: 28 Mar 1990
 Last Updated on STN: 28 Mar 1990
 Entered Medline: 20 Feb 1990

AB Gliosarcomas contain both neuro-ectodermal and mesenchymal elements. Its histogenesis has been much debated and endothelial and adventitial fibroblast origins have been suggested, as has a "histiocytic" origin following the demonstration of antiprotease activity. Eight gliosarcomas have been examined with a panel of ten monoclonal and polyclonal antibodies to investigate the origin of the sarcomatous element. Glial fibrillary acid protein expression showed a sharp distinction between gliomatous and sarcomatous tumour components. Contrary to some previous reports factor 8-related antigen and Ulex europeus agglutinin stained vascular luminal endothelium but no tumour cells. Vimentin and fibronectin expression was extensive and confined largely to sarcomatous areas. Desmin and neurofilament protein could not be demonstrated in any of the cases. Numerous cells, particularly in the sarcoma areas, expressed alpha-1-antitrypsin and -chymotrypsin. A proportion of these stained for the monocyte/macrophage marker MAC 387. Four cases focally exhibited a true storiform pattern and this and the immunohistochemical results suggest analogies with the fibrous histiocytomas. These tumours contain reactive histiocytes but are now thought to be derived from fibroblasts or from pluripotent mesenchymal cells in perivascular adventitia. This resembles the pattern exhibited in the sarcomatous component of gliosarcomas.

L9 ANSWER 4 OF 14 MEDLINE on STN
 ACCESSION NUMBER: 89361857 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2769483
 TITLE: Spindle cell and pleomorphic lipoma: an immunohistochemical study and histogenetic analysis.
 AUTHOR: Beham A; Schmid C; Hodl S; Fletcher C D
 CORPORATE SOURCE: Institute of Pathology, University of Graz Medical School,

Austria.
 SOURCE: The Journal of pathology, (1989 Jul) Vol. 158,
 No. 3, pp. 219-22.
 Journal code: 0204634. ISSN: 0022-3417.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198909
 ENTRY DATE: Entered STN: 9 Mar 1990
 Last Updated on STN: 9 Mar 1990
 Entered Medline: 29 Sep 1989

AB Twenty-two spindle cell lipomas and seven pleomorphic lipomas were investigated immunohistochemically in order to study the differentiation of the non-adipocytic elements. In all cases, neither spindle cells nor pleomorphic cells reacted with antibodies to a monocyte/macrophage antigen (MAC-387), fibronectin, laminin or type IV collagen. The absence of demonstrable basement membrane material argues against the possible prelipoblastic nature of these cells. With the antibody to S-100 protein, spindle cells were immunonegative, whereas pleomorphic cells sometimes revealed an intracytoplasmic weak to moderate staining reaction. In the light of what is known about the development of adipose tissue, our results would support the hypothesis of Bolen and Thorning (Am J Surg Pathol 1981; 5: 435-441) that spindle cell lipoma is composed of adipocytes and non-fat storing immature mesenchymal cells. It would appear that pleomorphic lipoma is similarly derived but that in some cases adipocytic differentiation is also abnormal. The characteristic clinical distribution of these two types of tumour may be of relevance in determining the cause of these unusual benign patterns of differentiation.

L9 ANSWER 5 OF 14 MEDLINE on STN
 ACCESSION NUMBER: 88059062 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 3500170
 TITLE: Modulation by recombinant interleukin 1 of synthesis of types I and III collagens and associated procollagen mRNA levels in cultured human cells.
 AUTHOR: Goldring M B; Krane S M
 CORPORATE SOURCE: Department of Medicine, Harvard Medical School, Boston, Massachusetts.
 CONTRACT NUMBER: AM-03490 (NIADDK)
 AM-03564 (NIADDK)
 AM-07258 (NIADDK)
 SOURCE: The Journal of biological chemistry, (1987 Dec 5)
 Vol. 262, No. 34, pp. 16724-9.
 Journal code: 2985121R. ISSN: 0021-9258.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198801
 ENTRY DATE: Entered STN: 5 Mar 1990
 Last Updated on STN: 3 Feb 1997
 Entered Medline: 12 Jan 1988

AB Interleukin 1 (IL-1), a monocyte product, exerts a range of biological effects on nonimmune cells such as fibroblasts and chondrocytes including stimulation of synthesis and release of prostaglandin E2 (PGE2) and collagenase. We have previously shown that crude mononuclear cell-conditioned medium, which contains IL-1, also stimulates synthesis of types I and III collagens by human synovial and dermal fibroblasts and chondrocytes when the formation of PGE2, which inhibits collagen synthesis, is blocked by indomethacin. To determine whether IL-1 is responsible for the affects observed using crude monocyte

-conditioned medium patterns of collagen synthesis in the three types of human cells in response to recombinant preparations of IL-1 were compared. Preincubation of chondrocytes or synovial fibroblasts with either murine (m)IL-1 alpha or human (h)IL-1 beta alone decreased synthesis of type I collagen and fibronectin. In contrast, when endogenous IL-1-stimulated PGE2 synthesis was blocked by indomethacin, an enhancing effect of IL-1 on synthesis of these matrix proteins was unmasked. The synthesis of type III collagen was enhanced by IL-1 to a greater extent than that of type I collagen in the presence of indomethacin. In human foreskin fibroblasts, which produced low levels of PGE2 even in the presence of IL-1, synthesis of types I and III collagens was increased by IL-1 either in the absence or presence of indomethacin. These cells were more responsive to the hIL-1 beta preparation than to the mIL-1 alpha (half-maximal stimulation of PGE2 production was observed at approximately 2.5-5 pM hIL-1 beta and at approximately 2.5 nM mIL-1 alpha). Levels of alpha 1 (I), alpha 2(I), and alpha 1(III) procollagen mRNAs measured by cytoplasmic dot hybridization paralleled the levels of collagens synthesized under the various experimental conditions. IL-1, therefore, is one product of monocytes capable of modulating collagen synthesis by these human mesenchymal cells probably by altering collagen gene expression. These studies suggest that both positive (IL-1) and negative (PGE2) signals may control collagen synthesis at the transcriptional level resulting in modulation of matrix turnover in cartilage, synovium, and skin.

L9 ANSWER 6 OF 14 MEDLINE on STN
 ACCESSION NUMBER: 85200262 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 3888308
 TITLE: The Mr 95,000 gelatin-binding protein in differentiated human macrophages and granulocytes.
 AUTHOR: Vartio T; Hedman K; Jansson S E; Hovi T
 SOURCE: Blood, (1985 May) Vol. 65, No. 5, pp. 1175-80.
 Journal code: 7603509. ISSN: 0006-4971.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 198506
 ENTRY DATE: Entered STN: 20 Mar 1990
 Last Updated on STN: 20 Mar 1990
 Entered Medline: 25 Jun 1985

AB Cultured adherent human macrophages and a promonocytic cell line, U 937, were previously shown to produce a Mr 95,000 gelatin-binding protein. The protein has no immunologic cross-reactivity with the well-characterized gelatin-binding protein fibronectin and the Mr 70,000 gelatin-binding protein produced by a variety of mesenchymal or epithelial cell types (T. Vartio et al, J Biol Chem 257:8862, 1982). In the present study the Mr 95,000 protein was found in Triton X-100 extracts of granulocytes purified from human blood buffy coat. The protein, as isolated by gelatin-agarose, was immunologically cross-reactive with the corresponding macrophage protein in immunoblotting assay. When peripheral blood and bone marrow cells were examined for the presence of the Mr 95,000 protein by indirect immunofluorescence, positive staining was detected only in differentiated granulocytes but not to any significant extent in metamyelocytes, myelocytes, promyelocytes, or in normal or leukemic blasts. In granulocytes the protein had a granular cytoplasmic distribution. In freshly prepared monocyte cultures, the Mr 95,000 protein was detected in low amounts in the cytoplasm, while along with differentiation of the cells into macrophages, the immunofluorescence increased in a reticular and vesicular cytoplasmic pattern and in a juxtanuclear cap, probably representing the Golgi complex. In conclusion, the Mr 95,000 gelatin-binding protein was specifically detected in macrophages and granulocytes and may thus serve as a differentiation

marker for these phagocytic cells.

L9 ANSWER 7 OF 14 MEDLINE on STN
ACCESSION NUMBER: 84001625 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6616317
TITLE: Identification of a bone matrix-derived chemotactic factor.
AUTHOR: Somerman M; Hewitt A T; Varner H H; Schiffmann E; Termine J; Reddi A H
SOURCE: Calcified tissue international, (1983 Jul) Vol. 35, No. 4-5, pp. 481-5.
Journal code: 7905481. ISSN: 0171-967X.
Report No.: NASA-84001625.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 198311
ENTRY DATE: Entered STN: 19 Mar 1990
Last Updated on STN: 19 Mar 1990
Entered Medline: 23 Nov 1983

AB When demineralized bone matrix powder is implanted subcutaneously in the rat, the early responses involve the appearance and proliferation of mesenchymal cells at the site of implantation, followed by cartilage and bone formation. The ability of cells to migrate to the implant suggests that chemotaxis may be a critical event in this process. Therefore, using the modified Boyden chamber assay, we tested extracts of demineralized bone matrix for chemotactic activity. We have identified and partially purified, on molecular sieve chromatography, a heat labile and trypsin-sensitive protein (Mr = 60,000-70,000) that is a potent chemoattractant for mouse calvaria, osteoblast-like cells (MMB-1), but not for monocytes (putative osteoclast precursors). These findings suggest that chemotactic protein(s) have a significant role in the recruitment of osteoprogenitor cells to a site of bone repair.

L9 ANSWER 8 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:431947 BIOSIS
DOCUMENT NUMBER: PREV200300431947
TITLE: hMSCs-host interactions in ectopic bone formation.
AUTHOR(S): Dodds, R. A. [Reprint Author]; Jennings, M. [Reprint Author]; Young, R. [Reprint Author]; Kostura, L. [Reprint Author]; Zhou, W. S.; Cooper, L. F.
CORPORATE SOURCE: Osiris Therapeutics, Baltimore, MD, USA
SOURCE: Journal of Bone and Mineral Research, (September 2002) Vol. 17, No. Suppl 1, pp. S340. print.
Meeting Info.: Twenty-Fourth Annual Meeting of the American Society for Bone and Mineral Research. San Antonio, Texas, USA. September 20-24, 2002. American Society for Bone and Mineral Research.
ISSN: 0884-0431 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Sep 2003
Last Updated on STN: 17 Sep 2003

L9 ANSWER 9 OF 14 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 92258851 EMBASE
DOCUMENT NUMBER: 1992258851
TITLE: Receptor-specific induction of insulin-like growth factor I in human monocytes by advanced glycosylation end product-modified proteins.
AUTHOR: Kirstein M.; Aston C.; Hintz R.; Vlassara H.
CORPORATE SOURCE: Picower Medical Research Institute, 350 Community

SOURCE: Drive, Manhasset, NY 11030, United States
Journal of Clinical Investigation, (1992) Vol. 90, No. 2,
pp. 439-446. .
ISSN: 0021-9738 CODEN: JCINAO

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20 Sep 1992
Last Updated on STN: 20 Sep 1992

AB Normal tissue homeostasis requires a finely balanced interaction between phagocytic scavenger cells (such as monocytes and macrophages) that degrade senescent material and mesenchymal cells (such as fibroblasts and smooth muscle cells), which proliferate and lay down new extracellular matrix. Macrophages and monocytes express specific surface receptors for advanced glycosylation end products (AGEs), which are covalently attached adducts resulting from a series of spontaneous nonenzymatic reactions of glucose with tissue proteins. Receptor-mediated uptake of AGE-modified proteins induces human monocytes to synthesize and release cytokines (TNF and IL-1), which are thought to contribute to normal tissue remodeling by mechanisms not entirely understood. We now report that AGEs also induce human monocytes to generate the potent progression growth factor insulin-like growth factor I (IGF-I), known to stimulate proliferation of mesenchymal cells. After in vitro stimulation with AGE-modified proteins, normal human blood monocytes express IGF-IA mRNA leading to the secretion of IGF-IA prohormone. The signal for IGF-IA mRNA induction seems to be initiated via the monocyte AGE-receptor, and to be propagated in an autocrine fashion via either IL-1 β or PDGF. These data introduce a novel regulatory system for IGF-I, with broad in vivo relevance, and provide an essential link to the chain of events leading from the spontaneously formed tissue AGEs, hypothesized to act as markers of protein senescence, to their replacement and to tissue remodeling by the locally controlled induction of growth factors.

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ACCESSION NUMBER: 90031995 EMBASE

DOCUMENT NUMBER: 1990031995

TITLE: Malignant fibrous histiocytoma is not related to histiocytes: Consistent phenotypic expression of CD13, CD10, and vimentin in four malignant fibrous histiocytoma cell lines.

AUTHOR: Hsu S.-M.; Rohol P.J.M.; Ko Y.H.; Lok M.-S.

CORPORATE SOURCE: Department of Pathology, University of Texas, Health Science Center, Houston, TX, United States

SOURCE: Cancer Journal, (1989) Vol. 2, No. 12, pp. 423-429. .
ISSN: 0765-7846 CODEN: CANJEI

COUNTRY: France

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy

016 Cancer

025 Hematology

LANGUAGE: English

SUMMARY LANGUAGE: French; Spanish; English

ENTRY DATE: Entered STN: 13 Dec 1991

Last Updated on STN: 13 Dec 1991

AB The authors studied four malignant fibrous histiocytoma (MFH) cell lines (MFH-1, MFH-2, GCT, MFHL) for their phenotypic expression by using a large panel of antibodies, including antibodies directed against monocytes/histiocytes and various types of intermediate filaments. Most MFH cells were positively stained by anti-vimentin, -CD13 (MY7), and -CD10 (J5) and were variably stained by anti-fibronectin,

cytokeratin, factor VIII, and HLA-Dr. They did not express CD45 (T200), desmin, muscle actin, myoglobulin, glial fibrillar antigen, neurofilaments, neuron-specific enolase, or markers associated with monocytes/histiocytes. The phenotype of MFH cells is similar, but not exclusive, to the phenotypes of cultured fibroblasts and of cells from fibrosarcomas. The cultured MFH cells did not exhibit sufficient phenotypic, ultrastructural, and functional features to be clarified as histiocytes. Treatment of cells with phorbol ester, retinoic acid, and fibroblast growth factor did not elicit any apparent differentiation toward histiocytes. The results indicate that these cells are related to mesenchymal cells rather than to histiocytes.

L9 ANSWER 11 OF 14 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:976796 SCISEARCH

THE GENUINE ARTICLE: 498QB

TITLE: Glucocorticoids down-regulate the extracellular matrix proteins fibronectin, fibulin-1 and fibulin-2 in bone marrow stroma

AUTHOR: Gu Y C; Talts J F; Gullberg D; Timpl R; Ekblom M (Reprint)

CORPORATE SOURCE: Lund Univ, Dept Cell & Mol Biol, BMC B12, SE-22184 Lund, Sweden (Reprint); Lund Univ, Dept Cell & Mol Biol, SE-22184 Lund, Sweden; Lund Univ, Dept Lab Med, SE-22184 Lund, Sweden; Univ Lund Hosp, S-22185 Lund, Sweden; Uppsala Univ, Dept Cell & Mol Biol, S-75148 Uppsala, Sweden; Max Planck Inst Biochem, D-8033 Martinsried, Germany

COUNTRY OF AUTHOR: Sweden; Germany

SOURCE: EUROPEAN JOURNAL OF HAEMATOLOGY, (SEP 2001) Vol. 67, No. 3, pp. 176-184. ISSN: 0902-4441.

PUBLISHER: MUNKSGAARD INT PUBL LTD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 56

ENTRY DATE: Entered STN: 21 Dec 2001

Last Updated on STN: 21 Dec 2001

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Glucocorticoids regulate hematopoietic cell interactions with the bone marrow microenvironment, but the molecules involved in the regulation are still largely unknown. We have studied the effect of glucocorticoids on mRNA expression and protein synthesis of the major extracellular matrix adhesion protein fibronectin and three other extracellular proteins, fibulin-1, fibulin-2 and nidogen-1, in mouse bone marrow cultures and in a hematopoiesis supporting the stromal MC3T3-G2/PA6 cell line. Glucocorticoids suppressed mRNA expression and protein synthesis of fibronectin, fibulin-1 and fibulin-2, but not nidogen-1, in adherent cells of bone marrow cultures, as shown by Northern blot analysis and immunoprecipitation. mRNA levels of all four proteins were down-regulated by dexamethasone in MC3T3-G2/PA6 cells, indicating a direct glucocorticoid effect on cells synthesizing extracellular matrix proteins. Dexamethasone down-regulated fibronectin mRNA rapidly, within 2 h of treatment, in the stromal cells. This effect did not require mRNA or protein synthesis, as shown by Northern blot analysis after treatment by actinomycin D and cycloheximide. Interferon- α , which also has been reported to modulate haematopoietic cell-matrix interactions, did not affect mRNA expression of the proteins in MC3T3-G2/PA6 cells. Our results indicate that glucocorticoids down-regulate expression of several mesenchymal-type extracellular matrix molecules in bone marrow, but with a variable effect on different proteins. Thus one mechanism by which glucocorticoids regulate haematopoiesis may be by altering the relative proportions of extracellular matrix proteins.

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ACCESSION NUMBER: 1998:936946 SCISEARCH

THE GENUINE ARTICLE: 145PT

TITLE: Living skin substitutes: Survival and function of fibroblasts seeded in a dermal substitute in experimental wounds

AUTHOR: Lamme E N (Reprint); van Leeuwen R T J; Jonker A; van Marle J; Middelkoop E

CORPORATE SOURCE: Univ Amsterdam, Acad Med Ctr, Dept Dermatol, Wound Healing Res Grp, Neurozintuigen Lab, K2N-210, POB 22700, NL-1100 DE Amsterdam, Netherlands (Reprint); Univ Amsterdam, Acad Med Ctr, Dept Dermatol, Wound Healing Res Grp, Neurozintuigen Lab, NL-1100 DE Amsterdam, Netherlands; Univ Amsterdam, Acad Med Ctr, Dept Cell Biol & Histol, NL-1100 DE Amsterdam, Netherlands; Univ Amsterdam, Acad Med Ctr, Dept Electron Microscopy, NL-1100 DE Amsterdam, Netherlands; Red Cross Hosp & Burns Fdn, Burns Ctr, Beverwijk, Netherlands

COUNTRY OF AUTHOR: Netherlands

SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (DEC 1998) Vol. 111, No. 6, pp. 989-995.. ISSN: 0022-202X.

PUBLISHER: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148 USA.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 52

ENTRY DATE: Entered STN: 1998

Last Updated on STN: 1998

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The healing of full-thickness skin defects requires extensive synthesis and remodeling of dermal and epidermal components. Fibroblasts play an important role in this process and are being incorporated in the latest generation of artificial dermal substitutes. We studied the fate of fibroblasts seeded in our artificial elastin/collagen dermal substitute and the influence of the seeded fibroblasts on cell migration and dermal substitute degradation after transplantation to experimental full-thickness wounds in pigs. Wounds were treated with either dermal substitutes seeded with autologous fibroblasts or acellular substitutes. Seeded fibroblasts, labeled with a PKH-26 fluorescent cell marker, were detected in the wounds with fluorescence microscopy and quantitated with flow cytofluorometric analysis of single-cell suspensions of wound tissue. The cellular infiltrate was characterized for the presence of mesenchymal cells (vimentin), monocytes/macrophages, and vascular cells. Dermal substitute degradation was quantitated by image analysis of wound sections stained with Herovici's staining. In the wounds treated with the seeded dermal substitute, fluorescent PKH-26-labeled cells were detectable up to 6 d and were positive for vimentin but not for the macrophage antibody. After 5 d, flow cytofluorometry showed the presence of $3.1 (+/- 0.9) \times 10^6$ (mean \pm SD, $n = 7$) PKH-26-positive cells in these wounds, whereas initially only 1×10^6 fluorescent fibroblasts had been seeded. In total, the percentage of: mesenchymal cells minus the macrophages was similar after 5 d between wounds treated with the seeded and the acellular substitutes. In the wounds treated with the seeded substitute, however, 19.5% of the mesenchymal cells were of seeded origin. Furthermore, the rate of substitute degradation in the seeded wounds was significantly lower at 2-4 wk after wounding than in wounds treated with the acellular substitute. Vascular in-growth and the number of infiltrated macrophages were not different. In conclusion, cultured dermal fibroblasts seeded in an artificial dermal substitute and transplanted onto full-thickness wounds in pigs survived and proliferated. The observed effects of seeded fibroblasts on dermal regeneration appeared to be mediated by reducing subcutaneous fibroblastic cell migration and/or proliferation into the

wounds without impairing migration of monocytes/macrophages and endothelial cells, Moreover, the degradation of the implanted dermal substitute was retarded, indicating a protective activity of the seeded fibroblasts.

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ACCESSION NUMBER: 1992:633775 SCISEARCH

THE GENUINE ARTICLE: JU865

TITLE: ROLE OF MULTIPOTENT FIBROBLASTS IN THE HEALING COLONIC MUCOSA OF RABBITS - ULTRASTRUCTURAL AND IMMUNOCYTOCHEMICAL STUDY

AUTHOR: MORI N (Reprint); DOI Y; HARA K; YOSHIZUKA M; OHSATO K; FUJIMOTO S

CORPORATE SOURCE: UNIV OCCUPAT & ENVIRONM HLTH, SCH MED, DEPT ANAT, 1-1 ISEIGAOKA, YAHATANISHI KU, KITAKYUSHU, FUKUOKA 807, JAPAN; UNIV OCCUPAT & ENVIRONM HLTH, SCH MED, DEPT SURG, KITAKYUSHU, FUKUOKA 807, JAPAN

COUNTRY OF AUTHOR: JAPAN

SOURCE: HISTOLOGY AND HISTOPATHOLOGY, (OCT 1992) Vol. 7, No. 4, pp. 583-590.
ISSN: 0213-3911.

PUBLISHER: F HERNANDEZ, PLAZA FUENSANTA 2-7 C, 30008 MURCIA, SPAIN.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 40

ENTRY DATE: Entered STN: 1994

Last Updated on STN: 1994

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Light- and electron microscopy and immunocytochemistry were used to study the healing colonic mucosa of rabbits after experimental excision.

Between 3 and 5 days, abundant young fibroblasts which retained many features of mesenchymal cells invaded the growing capillaries into the loose connective tissue of the healing colonic mucosa. Our electron microscopy revealed the transformation of these young fibroblasts into smooth muscle cells, into histiocyte-like cells involved in phagocytotic activity, and into vasoformative cells incorporated into the growing capillaries. The mitotic proliferation of pre-existing smooth muscle cells at the ulcer margin did not seem to be the major reason for re-establishment of the muscular tissue.

The present immunocytochemistry revealed an active production of fibronectin in rough endoplasmic reticulum in the young fibroblasts. This may mean that this glycoprotein is involved in the re-establishment of both connective and muscular tissues by enhancement of adhesion and chemoattractant activities of such cells. In addition, the immunoreaction of endothelial cells of the growing capillaries suggests a role of this glycoprotein in the acceleration of the neocapillarization.

L9 ANSWER 14 OF 14 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1992:440922 SCISEARCH

THE GENUINE ARTICLE: JD972

TITLE: DISTRIBUTION OF THE EXTRACELLULAR-MATRIX PROTEINS TENASCIN, FIBRONECTIN, AND VITRONECTIN IN FETAL, INFANT, AND ADULT HUMAN SPLEENS

AUTHOR: LIAKKA K A (Reprint); AUTIOHARMAINEN H I

CORPORATE SOURCE: UNIV OULU, DEPT PATHOL, KAJAANINTIE 52 D, SF-90220 OULU 22, FINLAND (Reprint)

COUNTRY OF AUTHOR: FINLAND

SOURCE: JOURNAL OF HISTOCHEMISTRY & CYTOCHEMISTRY, (AUG 1992) Vol. 40, No. 8, pp. 1203-1210.
ISSN: 0022-1554.

PUBLISHER: HISTOCHEMICAL SOC INC, MT SINAI MEDICAL CENTER 19 EAST

98TH ST SUTIE 9G, NEW YORK, NY 10029.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 43
ENTRY DATE: Entered STN: 1994
Last Updated on STN: 1994

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Using immunohistochemistry, we investigated the distribution of the extracellular matrix (ECM) glycoproteins tenascin, fibronectin, and vitronectin in fetal [16-24 gestational weeks (GW)], infant (40 GW), and adult human spleens to clarify the presence of these proteins during different phases of maturation. In the red and white pulp, tenascin and fibronectin were constant components of the reticular fibers from the age of 18 GW onwards, whereas vitronectin was seen only in adult spleens. The immunohistochemical staining patterns of tenascin and fibronectin remained unchanged at different fetal ages. Ring fibers, which are modified basement membranes around venous sinuses, became visible relatively late, and in adult spleens they contained both tenascin and vitronectin but lacked fibronectin. The composition of the ring fibers is therefore clearly different from that of ordinary basement membranes, which have not been reported to contain tenascin or vitronectin. The rapidly increasing number of reticular fibers in the spleen at the age of approximately 18 GW corresponds with the beginning of lymphatic colonization. Reticular fibers, rich in ECM glycoproteins, form a framework to which cells can migrate and attach. We suggest that the composition of these fibers might be important for lymphatic colonization and function of the spleen.

=> s monocyte phenotype

L10 247 MONOCYTE PHENOTYPE

=> s mesenchymal near2 differentiona

L11 0 MESENCHYMAL NEAR2 DIFFERENTIONA

=> s mesenchymal near2 differentiation

L12 0 MESENCHYMAL NEAR2 DIFFERENTION

=> s mesenchymal differentiation

L13 0 MESENCHYMAL DIFFERENTIONA

=> s mesenchymal and differentiation

L14 4 MESENCHYMAL AND DIFFERENTION

=> s l14 and fibronectin

L15 0 L14 AND FIBRONECTIN

=> s monocyte (P) MULTIPOTENT

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'MONOCYTE (P) MULTIPOTE'

L16 349 MONOCYTE (P) MULTIPOTENT

=> S L16 (P) MESENCHYMAL

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'L111 (P) MESENCHYM'

L17 5 L16 (P) MESENCHYMAL

=> S L16 AND FIBRONECTIN

L18 1 L16 AND FIBRONECTIN

=> DISP L18 IBIB ABS 1-1

L18 ANSWER 1 OF 1 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on

STN

ACCESSION NUMBER: 1994:306415 SCISEARCH
THE GENUINE ARTICLE: NM598
TITLE: EXPRESSION OF ADHESION MOLECULES ON HUMAN HEMATOPOIETIC
PROGENITOR CELLS AT DIFFERENT MATURATIONAL STAGES
AUTHOR: KOBAYASHI M (Reprint); IMAMURA M; UEDE T; SAKURADA K;
MAEDA S; IWASAKI H; TSUDA Y; MUSASHI M; MIYAZAKI T
CORPORATE SOURCE: HOKKAIDO UNIV, SCH MED, INST CANC, PATHOL LAB, NISHI-7,
KITA-15, KITA KU, SAPPORO, HOKKAIDO 060, JAPAN (Reprint);
HOKKAIDO UNIV, SCH MED, DEPT INTERNAL MED 3, SAPPORO,
HOKKAIDO 060, JAPAN; HOKKAIDO UNIV, INST IMMUNOL SCI,
IMMUNOPATHOGENESIS SECT, SAPPORO, HOKKAIDO 060, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: STEM CELLS, (MAY 1994) Vol. 12, No. 3, pp. 316-321.
ISSN: 1066-5099.
PUBLISHER: ALPHAMED PRESS, 4100 S KETTERING BLVD, DAYTON, OH
45439-2092.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 24
ENTRY DATE: Entered STN: 1994
Last Updated on STN: 1994

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In this report we examined the expression of several adhesion
molecules on human hematopoietic progenitor cells at different
maturational stages. Human hematopoietic progenitor cell-enriched
fractions were prepared from bone marrow cells by depleting lymphocytes
and monocytes (CD2+, CD14+ and CD19+ cells). These cells were
separated into adhesion molecule-positive and -negative cell populations
by immunomagnetic separation methods and then assessed for their ability
to form various colony forming cells (CFC). CD44 and CD49d were expressed
on multipotent hematopoietic progenitor cells, or mixed colony
forming units (CFU-Mix), erythroid burst forming units (BFU-E),
granulocyte-macrophage CFU (CFU-GM) and erythroid CFU (CFU-E). Leu8 was
expressed on CFU-Mix, BFU-E and some populations of CFU-GM, but not CFU-E.
CD11a was expressed on some populations of CFU-Mix, CFU-GM and BFU-E.
CD54 was expressed only on some populations of CFU-GM. These results
suggest that Leu8, CD44, CD49d and CD11a appear to play important roles in
the differentiation and proliferation of human hematopoietic progenitor
cells at different maturational stages in the bone marrow
microenvironment.

=> S L16 AND CD14

L19 18 L16 AND CD14

=> S L16 AND CD34

L20 49 L16 AND CD34

=> S L16 AND CD45

L21 7 L16 AND CD45

=> S L16 AND TYPE(A) I COLLAGEN

L22 0 L16 AND TYPE(A) I COLLAGEN

=> S MONOCYTE (P) FIBRONECTIN

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'MONOCYTE (P) FIBRONECT'

L23 2801 MONOCYTE (P) FIBRONECTIN

=> S L23 (P) MESENCHYMAL

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'L160 (P) MESENCHYM'

L24 41 L23 (P) MESENCHYMAL

=> DUP REM 41
ENTER L# LIST OR (END):END

=> DUP REM L24
PROCESSING COMPLETED FOR L24
L25 17 DUP REM L24 (24 DUPLICATES REMOVED)

=> S L25 AND PY<2003
1 FILES SEARCHED...
5 FILES SEARCHED...
L26 8 L25 AND PY<2003

=> DISP L26 IBIB ABS 1-8

L26 ANSWER 1 OF 8 MEDLINE on STN
ACCESSION NUMBER: 97459730 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9315690
TITLE: Integrin-mediated stimulation of monocyte chemotactic protein-1 expression.
AUTHOR: Marra F; Pastacaldi S; Romanelli R G; Pinzani M; Ticali P; Carloni V; Laffi G; Gentilini P
CORPORATE SOURCE: Istituto di Medicina Interna, Universita di Firenze, Florence, Italy.. f.marra@dfc.unifi.it
SOURCE: FEBS letters, (1997 Sep 8) Vol. 414, No. 2, pp. 221-5.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 5 Nov 1997
Last Updated on STN: 5 Nov 1997
Entered Medline: 21 Oct 1997

AB We investigated whether activation of integrin receptors could modulate the expression of monocyte chemotactic protein-1 (MCP-1) in human hepatic stellate cells (HSC), mesenchymal cells responsible for extracellular matrix synthesis within the liver. When compared to non-adherent cells, HSC plated on collagen types I or IV, or fibronectin, showed increased MCP-1 gene expression and protein secretion in the conditioned medium. Increased MCP-1 secretion was also observed when cells were plated on dishes coated with a monoclonal antibody directed against the beta1-integrin subunit, demonstrating that ligation of beta1-integrins is sufficient to stimulate MCP-1 expression. Conversely, integrin-independent cell adhesion on poly-L-lysine did not modify MCP-1 secretion. Disruption of the actin cytoskeleton by cytochalasin D blocked the collagen-dependent increase in MCP-1 secretion. Chemotactic assay of HSC-conditioned medium showed that HSC plated on collagen secrete higher amounts of chemotactic factors for lymphomonocytes, and that MCP-1 accounts for the great majority of this effect. These findings indicate a novel mechanism of MCP-1 regulation possibly relevant in those conditions where HSC interact with an altered extracellular matrix.

L26 ANSWER 2 OF 8 MEDLINE on STN
ACCESSION NUMBER: 93112431 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8417761
TITLE: In vitro effects of pentoxifylline on smooth muscle cell migration and blood monocyte production of chemotactic activity for smooth muscle cells: potential therapeutic

benefit in the adult respiratory distress syndrome.
 AUTHOR: Kullmann A; Vaillant P; Muller V; Martinet Y; Martinet N
 CORPORATE SOURCE: INSERM U. 14, CLERC Poumon, CHU, Nancy-Vandoeuvre, France.
 SOURCE: American journal of respiratory cell and molecular biology,
 (1993 Jan) Vol. 8, No. 1, pp. 83-8.
 Journal code: 8917225. ISSN: 1044-1549.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199302
 ENTRY DATE: Entered STN: 19 Feb 1993
 Last Updated on STN: 19 Feb 1993
 Entered Medline: 3 Feb 1993

AB The adult respiratory distress syndrome (ARDS) is a severe lung condition characterized by an acute lung injury leading to a massive intra-alveolar fibrosis with rapid lung failure. ARDS intra-alveolar fibrosis results from the migration of mesenchymal cells (mainly smooth muscle cells [SMC]) into the alveoli through alveolar epithelial basement membrane gaps resulting from the injury. SMC migration is followed by their replication and production of extracellular matrix, which leads to fibrosis. Thus, any pharmacologic agent able to prevent SMC migration should prevent, at least in part, intra-alveolar fibrosis. SMC migration is thought to be due to the presence, in the alveolar spaces, of chemotactic factors for mesenchymal cells, such as fibronectin and platelet-derived growth factor (PDGF). The local presence of these chemotactic factors can be due to plasmatic leakage, platelet degranulation, and mononuclear phagocyte activation. Pentoxifylline is a methylxanthine interacting with the biology of several types of cells, including red blood cells, neutrophils, blood monocytes, and endothelial cells. Pentoxifylline prescription has been suggested in ARDS with respect to its activity on neutrophils, its inhibition of tumor necrosis factor-alpha (TNF) release by mononuclear phagocytes, and its prevention of TNF-induced lung injury. Since pentoxifylline can modulate the migration of several cell types, we hypothesized that it could interfere with mesenchymal cell migration. SMC migratory response was measured in vitro with modified Boyden chemotactic chambers in the presence of PDGF, fibronectin, "platelet extract," and activated blood monocyte supernatants. Pentoxifylline, at therapeutic levels, significantly reduced SMC migration in response to the presence of these chemotactic activities. (ABSTRACT TRUNCATED AT 250 WORDS)

L26 ANSWER 3 OF 8 MEDLINE on STN
 ACCESSION NUMBER: 90118782 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2609937
 TITLE: Gliosarcoma: an immunohistochemical study.
 AUTHOR: Grant J W; Steart P V; Aguzzi A; Jones D B; Gallagher P J
 CORPORATE SOURCE: Institut fur Pathologie, Universitats-Krankenhaus, Zurich, Switzerland.
 SOURCE: Acta neuropathologica, (1989) Vol. 79, No. 3, pp. 305-9.
 Journal code: 0412041. ISSN: 0001-6322.
 PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199002
 ENTRY DATE: Entered STN: 28 Mar 1990
 Last Updated on STN: 28 Mar 1990
 Entered Medline: 20 Feb 1990

AB Gliosarcomas contain both neuro-ectodermal and mesenchymal elements. Its histogenesis has been much debated and endothelial and

adventitial fibroblast origins have been suggested, as has a "histiocytic" origin following the demonstration of antiprotease activity. Eight gliosarcomas have been examined with a panel of ten monoclonal and polyclonal antibodies to investigate the origin of the sarcomatous element. Glial fibrillary acid protein expression showed a sharp distinction between gliomatous and sarcomatous tumour components. Contrary to some previous reports factor 8-related antigen and Ulex europeus agglutinin stained vascular luminal endothelium but no tumour cells. Vimentin and fibronectin expression was extensive and confined largely to sarcomatous areas. Desmin and neurofilament protein could not be demonstrated in any of the cases. Numerous cells, particularly in the sarcoma areas, expressed alpha-1-antitrypsin and -chymotrypsin. A proportion of these stained for the monocyte/macrophage marker MAC 387. Four cases focally exhibited a true storiform pattern and this and the immunohistochemical results suggest analogies with the fibrous histiocytomas. These tumours contain reactive histiocytes but are now thought to be derived from fibroblasts or from pluripotent mesenchymal cells in perivascular adventitia. This resembles the pattern exhibited in the sarcomatous component of gliosarcomas.

L26 ANSWER 4 OF 8 MEDLINE on STN
 ACCESSION NUMBER: 89361857 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2769483
 TITLE: Spindle cell and pleomorphic lipoma: an immunohistochemical study and histogenetic analysis.
 AUTHOR: Beham A; Schmid C; Hodl S; Fletcher C D
 CORPORATE SOURCE: Institute of Pathology, University of Graz Medical School, Austria.
 SOURCE: The Journal of pathology, (1989 Jul) Vol. 158, No. 3, pp. 219-22.
 Journal code: 0204634. ISSN: 0022-3417..
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198909
 ENTRY DATE: Entered STN: 9 Mar 1990
 Last Updated on STN: 9 Mar 1990
 Entered Medline: 29 Sep 1989

AB Twenty-two spindle cell lipomas and seven pleomorphic lipomas were investigated immunohistochemically in order to study the differentiation of the non-adipocytic elements. In all cases, neither spindle cells nor pleomorphic cells reacted with antibodies to a monocyte/macrophage antigen (MAC-387), fibronectin, laminin or type IV collagen. The absence of demonstrable basement membrane material argues against the possible prelipoblastic nature of these cells. With the antibody to S-100 protein, spindle cells were immunonegative, whereas pleomorphic cells sometimes revealed an intracytoplasmic weak to moderate staining reaction. In the light of what is known about the development of adipose tissue, our results would support the hypothesis of Bolen and Thorning (Am J Surg Pathol 1981; 5: 435-441) that spindle cell lipoma is composed of adipocytes and non-fat storing immature mesenchymal cells. It would appear that pleomorphic lipoma is similarly derived but that in some cases adipocytic differentiation is also abnormal. The characteristic clinical distribution of these two types of tumour may be of relevance in determining the cause of these unusual benign patterns of differentiation.

L26 ANSWER 5 OF 8 MEDLINE on STN
 ACCESSION NUMBER: 88059062 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 3500170
 TITLE: Modulation by recombinant interleukin 1 of synthesis of types I and III collagens and associated procollagen mRNA

levels in cultured human cells.

AUTHOR: Goldring M B; Krane S M
CORPORATE SOURCE: Department of Medicine, Harvard Medical School, Boston, Massachusetts.
CONTRACT NUMBER: AM-03490 (NIADDK)
AM-03564 (NIADDK)
AM-07258 (NIADDK)
SOURCE: The Journal of biological chemistry, (1987 Dec 5)
Vol. 262, No. 34, pp. 16724-9.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198801
ENTRY DATE: Entered STN: 5 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 12 Jan 1988

AB Interleukin 1 (IL-1), a monocyte product, exerts a range of biological effects on nonimmune cells such as fibroblasts and chondrocytes including stimulation of synthesis and release of prostaglandin E2 (PGE2) and collagenase. We have previously shown that crude mononuclear cell-conditioned medium, which contains IL-1, also stimulates synthesis of types I and III collagens by human synovial and dermal fibroblasts and chondrocytes when the formation of PGE2, which inhibits collagen synthesis, is blocked by indomethacin. To determine whether IL-1 is responsible for the affects observed using crude monocyte-conditioned medium patterns of collagen synthesis in the three types of human cells in response to recombinant preparations of IL-1 were compared. Preincubation of chondrocytes or synovial fibroblasts with either murine (m) IL-1 alpha or human (h) IL-1 beta alone decreased synthesis of type I collagen and fibronectin. In contrast, when endogenous IL-1-stimulated PGE2 synthesis was blocked by indomethacin, an enhancing effect of IL-1 on synthesis of these matrix proteins was unmasked. The synthesis of type III collagen was enhanced by IL-1 to a greater extent than that of type I collagen in the presence of indomethacin. In human foreskin fibroblasts, which produced low levels of PGE2 even in the presence of IL-1, synthesis of types I and III collagens was increased by IL-1 either in the absence or presence of indomethacin. These cells were more responsive to the hIL-1 beta preparation than to the mIL-1 alpha (half-maximal stimulation of PGE2 production was observed at approximately 2.5-5 pM hIL-1 beta and at approximately 2.5 nM mIL-1 alpha). Levels of alpha 1 (I), alpha 2(I), and alpha 1(III) procollagen mRNAs measured by cytoplasmic dot hybridization paralleled the levels of collagens synthesized under the various experimental conditions. IL-1, therefore, is one product of monocytes capable of modulating collagen synthesis by these human mesenchymal cells probably by altering collagen gene expression. These studies suggest that both positive (IL-1) and negative (PGE2) signals may control collagen synthesis at the transcriptional level resulting in modulation of matrix turnover in cartilage, synovium, and skin.

L26 ANSWER 6 OF 8 MEDLINE on STN
ACCESSION NUMBER: 85200262 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3888308
TITLE: The Mr 95,000 gelatin-binding protein in differentiated human macrophages and granulocytes.
AUTHOR: Vartio T; Hedman K; Jansson S E; Hovi T
SOURCE: Blood, (1985 May) Vol. 65, No. 5, pp. 1175-80.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198506
ENTRY DATE: Entered STN: 20 Mar 1990
Last Updated on STN: 20 Mar 1990
Entered Medline: 25 Jun 1985

AB Cultured adherent human macrophages and a promonocytic cell line, U 937, were previously shown to produce a Mr 95,000 gelatin-binding protein. The protein has no immunologic cross-reactivity with the well-characterized gelatin-binding protein fibronectin and the Mr 70,000 gelatin-binding protein produced by a variety of mesenchymal or epithelial cell types (T. Vartio et al, J Biol Chem 257:8862, 1982). In the present study the Mr 95,000 protein was found in Triton X-100 extracts of granulocytes purified from human blood buffy coat. The protein, as isolated by gelatin-agarose, was immunologically cross-reactive with the corresponding macrophage protein in immunoblotting assay. When peripheral blood and bone marrow cells were examined for the presence of the Mr 95,000 protein by indirect immunofluorescence, positive staining was detected only in differentiated granulocytes but not to any significant extent in metamyelocytes, myelocytes, promyelocytes, or in normal or leukemic blasts. In granulocytes the protein had a granular cytoplasmic distribution. In freshly prepared monocyte cultures, the Mr 95,000 protein was detected in low amounts in the cytoplasm, while along with differentiation of the cells into macrophages, the immunofluorescence increased in a reticular and vesicular cytoplasmic pattern and in a juxtannuclear cap, probably representing the Golgi complex. In conclusion, the Mr 95,000 gelatin-binding protein was specifically detected in macrophages and granulocytes and may thus serve as a differentiation marker for these phagocytic cells.

L26 ANSWER 7 OF 8 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 90031995 EMBASE

DOCUMENT NUMBER: 1990031995

TITLE: Malignant fibrous histiocytoma is not related to histiocytes: Consistent phenotypic expression of CD13, CD10, and vimentin in four malignant fibrous histiocytoma cell lines.

AUTHOR: Hsu S.-M.; Rohol P.J.M.; Ko Y.H.; Lok M.-S.

CORPORATE SOURCE: Department of Pathology, University of Texas, Health Science Center, Houston, TX, United States

SOURCE: Cancer Journal, (1989) Vol. 2, No. 12, pp. 423-429. .
ISSN: 0765-7846 CODEN: CANJEI

COUNTRY: France

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
025 Hematology

LANGUAGE: English

SUMMARY LANGUAGE: French; Spanish; English

ENTRY DATE: Entered STN: 13 Dec 1991

Last Updated on STN: 13 Dec 1991

AB The authors studied four malignant fibrous histiocytoma (MFH) cell lines (MFH-1, MFH-2, GCT, MFHL) for their phenotypic expression by using a large panel of antibodies, including antibodies directed against monocytes/histiocytes and various types of intermediate filaments. Most MFH cells were positively stained by anti-vimentin, -CD13 (MY7), and -CD10 (J5) and were variably stained by anti-fibronectin, cytokeratin, factor VIII, and HLA-Dr. They did not express CD45 (T200), desmin, muscle actin, myoglobulin, glial fibrillary antigen, neurofilaments, neuron-specific enolase, or markers associated with monocytes/histiocytes. The phenotype of MFH cells is similar, but not exclusive, to the phenotypes of cultured fibroblasts and of cells from fibrosarcomas. The cultured MFH cells did not exhibit sufficient phenotypic,

ultrastructural, and functional features to be clarified as histiocytes. Treatment of cells with phorbol ester, retinoic acid, and fibroblast growth factor did not elicit any apparent differentiation toward histiocytes. The results indicate that these cells are related to mesenchymal cells rather than to histiocytes.

L26 ANSWER 8 OF 8 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1992:22258850 BIOTECHNO

TITLE: Receptor-specific induction of insulin-like growth factor I in human monocytes by advanced glycosylation end product-modified proteins

AUTHOR: Kirstein M.; Aston C.; Hintz R.; Vlassara H.

CORPORATE SOURCE: Picower Medical Research Institute, 350 Community Drive, Manhasset, NY 11030, United States.

SOURCE: Journal of Clinical Investigation, (1992), 90/2 (439-446)

CODEN: JCINAO ISSN: 0021-9738

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 1992:22258850 BIOTECHNO

AB Normal tissue homeostasis requires a finely balanced interaction between phagocytic scavenger cells (such as monocytes and macrophages) that degrade senescent material and mesenchymal cells (such as fibroblasts and smooth muscle cells), which proliferate and lay down new extracellular matrix. Macrophages and monocytes express specific surface receptors for advanced glycosylation end products (AGEs), which are covalently attached adducts resulting from a series of spontaneous nonenzymatic reactions of glucose with tissue proteins. Receptor-mediated uptake of AGE-modified proteins induces human monocytes to synthesize and release cytokines (TNF and IL-1), which are thought to contribute to normal tissue remodeling by mechanisms not entirely understood. We now report that AGEs also induce human monocytes to generate the potent progression growth factor insulin-like growth factor I (IGF-I), known to stimulate proliferation of mesenchymal cells. After in vitro stimulation with AGE-modified proteins, normal human blood monocytes express IGF-IA mRNA leading to the secretion of IGF-IA prohormone. The signal for IGF-IA mRNA induction seems to be initiated via the monocyte AGE-receptor, and to be propagated in an autocrine fashion via either IL-1 β or PDGF. These data introduce a novel regulatory system for IGF-I, with broad in vivo relevance, and provide an essential link to the chain of events leading from the spontaneously formed tissue AGEs, hypothesized to act as markers of protein senescence, to their replacement and to tissue remodeling by the locally controlled induction of growth factors.

=> S FIBRONECTIN(A) COAT?

L27 3603 FIBRONECTIN(A) COAT?

=> S MONOCYTE (P) MESENCHYMAL

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'MONOCYTE (P) MESENCHYM'

L28 1055 MONOCYTE (P) MESENCHYMAL

=> L27 AND L28

L27 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.

For a list of commands available to you in the current file, enter

"HELP COMMANDS" at an arrow prompt (=>).

=> S L27 AND L28

L29 3 L27 AND L28

=> DUP REM L29
PROCESSING COMPLETED FOR L29
L30 3 DUP REM L29 (0 DUPLICATES REMOVED)

=> S L30 <2003
NUMERIC EXPRESSION NOT VALID 'L205 <2003'
Numeric search expressions contain an operator (=,>,<,<=>,<=>), a field
qualifier, and the number or a range to be searched. Examples of
valid expressions are 'LD>6', '260-280/MW', and '10 < LD < 30'. For a
list of field codes in the current file, enter "HELP SFIELDS" at an
arrow prompt (=>). For more information on searching in numeric
fields, enter "HELP NUMERIC".

=> S L30 AND PY<2004
1 FILES SEARCHED...
4 FILES SEARCHED...
L31 2 L30 AND PY<2004

=> DISP L31 IBIB ABS 1-2

L31 ANSWER 1 OF 2 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:154851 BIOSIS
DOCUMENT NUMBER: PREV200400148386
TITLE: Standardization of a colony assay to further characterize
endothelial precursor cells in blood and bone marrow.
AUTHOR(S): Faulkes, Sharlene M. [Reprint Author]; Pereira, Carla
[Reprint Author]; Peters, Carrie E. [Reprint Author];
Thomas, Terry E. [Reprint Author]; Eaves, Allen C. [Reprint
Author]; Emer, Clarke [Reprint Author]
CORPORATE SOURCE: StemCell Technologies, Inc., Vancouver, BC, Canada
SOURCE: Blood, (November 16 2003) Vol. 102, No. 11, pp.
827a. print.
Meeting Info.: 45th Annual Meeting of the American Society
of Hematology. San Diego, CA, USA. December 06-09, 2003.
American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004

AB Circulating endothelial precursor cells (EPCs) are thought to contribute
to the maintenance and repair of heart tissue and may be useful in
treating myocardial infarcts. Mobilization of EPCs from the bone marrow
or the injection of culture expanded mesenchymal cells are two
possible means of targeting EPCs to the site of injury. However, little
is known about optimal protocols to mobilize endothelial cells and it is
not known whether culture expanded mesenchymal cells will
produce the desired functional effects in vivo. It has been difficult to
phenotypically identify the cells capable of promoting heart tissue repair
as EPCs express the same cell surface markers as subsets of hematopoietic
cells. A recently described colony assay for EPCs showed an inverse
correlation between the number of circulating colony-forming cells (CFCs)
and the risk of cardiovascular-disease (Hill et al, NEJM 348(7):2003).
Based on this indirect evidence that the precursor cells which read out in
this colony assay play a role in cardiac tissue maintenance and repair, we
proceeded to characterize these CFC in normal peripheral blood (PB) and
bone marrow (BM). Mononuclear cells from PB and BM were isolated by
Ficoll density-gradient centrifugation and plated on fibronectin
coated 6 well plates (5X10⁶ cells per well) in liquid medium
containing selected fetal bovine serum (EndoCultTM medium, StemCell
Technologies). After 48 hours, the nonadherent cells were collected and

1X10⁶ cells (PB) or 5X10⁵ cells (BM) were plated in replicate fibronectin coated 24 well plates. CFCs were quantitated microscopically 5 days later, where a colony was defined as a central core of "round" cells with more elongated "sprouting" cells at the periphery. The frequency of CFCs in PB (1: 75,000+-20,000, n=10) was lower than that of BM (1:10,000+-2500, n=5). Confirmation that these colonies were of the endothelial lineage was provided by immunostaining with antibodies to markers expressed by mature endothelial cells (e.g., vWF). To identify the cell populations removed during the first 48 hours of adherence on fibronectin coated plates, flow cytometric analysis was performed on 5 consecutive BM samples prior to and following adherence. Incubation on fibronectin coated plates removed 31.8+-0.5% of mononuclear cells including monocytes (CD14+) and mature endothelial cells (CD105+/CD45-). All detectable CD146 cells also expressed CD3 and CD45 indicating that they were activated T cells; these cells were not depleted. In summary, this colony assay allows the quantitation of EPCs which importantly may play a role in cardiac tissue maintenance and repair. This assay provides a standardized tool for researchers to study endothelial cell biology, and may facilitate assessment of the risk of cardiovascular disease in asymptomatic individuals.

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AB Hematopoietic stem cells (HSC), Marrow stromal cells or Mesenchymal stem cells (MSC) in adult bone marrow are originated from mesoderm in developmental status. HSC have been reported to give rise to all types of blood cells and to be maintained by several kinds of cytokines and extrinsic factors from MSC. However, the developmental relationship between HSC and MSC are still unclear. We hypothesize that purified HSC may have multi-potential to differentiate into not only hematopoietic cells, but also other kinds of connective tissue cells such as adipocytes or osteocytes. To isolate pure HSC, we utilized dual-wavelength flow cytometric analysis with the fluorescent DNA-binding dye Hoechst 33342 and combined the staining of cell surface markers: CD34, c-Kit, Sca-1, Lineage markers (CD3, B220, Mac-1, Gr-1, Ter119). The highest dye efflux (or lowest staining) "Tip"-Side Population (SP) cells were positive for c-Kit (>98%, n>30) and mostly negative for lineage markers (Lin: <2%, n>30), and overlapped with the CD34 negative c-Kit positive Sca-1 positive Lineage negative (CD34- KSL) population with a ratio of 20%. By this method, we isolated a single CD34- KSL "Tip" -SP cells from BM of 8- to 10-week-old transgenic mice that constitutively express enhanced green fluorescence protein (EGFP) in all tissue, driven by the CAG promoter, by fluorescence-activated cell sorting (FACS). To

generate chimeric mice, we transplanted a single CD34- KSL Tip-SP cell into lethally irradiated 8- to 10-week-old wild type C57/BL6 mice. The donor cells derived from a single EGFP+ CD34- KSL Tip-SP cell were detectable (>1%) in 70 out of 73 recipients (95.9%) of the host animals 12 weeks after transplantation. All lineages of hematopoietic cells, including myelo/monocytes, lymphocytes, and erythrocytes, -derived from a single "Tip"-SP cell were detectable in each recipients for longer than 6 months after transplantation. To investigate the differentiation potency to mesenchymal cell lineage, we cultured 1X10⁵ whole BM cells from engrafted recipients with 80% donor origin EGFP-positive cells, on the fibronectin coated 96-well dishes. 14 days after cultivation, the spindle shaped adherent cells were detected and stained with anti-GFP, anti-CD45 (pan-hematopoietic marker), and Hoechst by immunohistochemistry method. GFP+ adherent cells were detected 0.83% of Hoechst positive cells and GFP+ CD45- mononuclear spindle shaped adherent cells were detected 0.03% of Hoechst positive cells. Then we differentiated these adherent cells in the adipocytes or osteocytes tissue specific inducing medium for another 14 days. The adipocytes or osteocytes were all GFP negative recipient derived cells. Taken all, it is suggested that purified HSC rarely differentiated into MSC even originated from same mesoderm. Therefore, the purified HSC might be already committed to hematopoietic lineage and transdifferentiation to MSC occurs very low frequency.

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